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NEWS	1		Web Page for STN Seminar Schedule - N. America
NEWS	2	MAY 01	New CAS web site launched
NEWS	3	MAY 08	CA/CAplus Indian patent publication number format defined
NEWS	4	MAY 14	RDISCLOSURE on STN Easy enhanced with new search and display fields
NEWS	5	MAY 21	BIOSIS reloaded and enhanced with archival data
NEWS	6	MAY 21	TOXCENTER enhanced with BIOSIS reload
NEWS	7	MAY 21	CA/CAplus enhanced with additional kind codes for German patents
NEWS	8	MAY 22	CA/CAplus enhanced with IPC reclassification in Japanese patents
NEWS	9	JUN 27	CA/CAplus enhanced with pre-1967 CAS Registry Numbers
NEWS	10	JUN 29	STN Viewer now available
NEWS	11	JUN 29	STN Express, Version 8.2, now available
NEWS	12	JUL 02	LEMBASE coverage updated
NEWS	13	JUL 02	LMEDLINE coverage updated
NEWS	14	JUL 02	SCISEARCH enhanced with complete author names
NEWS	15	JUL 02	CHEMCATS accession numbers revised
NEWS	16	JUL 02	CA/CAplus enhanced with utility model patents from China
NEWS	17	JUL 16	CAplus enhanced with French and German abstracts
NEWS	18	JUL 18	CA/CAplus patent coverage enhanced
NEWS	19	JUL 26	USPATFULL/USPAT2 enhanced with IPC reclassification
NEWS	20	JUL 30	USGENE now available on STN
NEWS	21	AUG 06	CAS REGISTRY enhanced with new experimental property tags
NEWS	22	AUG 06	BEILSTEIN updated with new compounds
NEWS	23	AUG 06	FSTA enhanced with new thesaurus edition
NEWS	24	AUG 13	CA/CAplus enhanced with additional kind codes for granted patents
NEWS	25	AUG 20	CA/CAplus enhanced with CAS indexing in pre-1907 records

NEWS EXPRESS 29 JUNE 2007: CURRENT WINDOWS VERSION IS V8.2,
CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
AND CURRENT DISCOVER FILE IS DATED 05 JULY 2007.

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=> File .gerry2MBCE
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                                                          ENTRY    SESSION
FULL ESTIMATED COST                                        0.21       0.21

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=> S MIP-4 (L)CCRL2
L1            1 MIP-4 (L) CCRL2

=> D ibib Abs l1

L1   ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER:         2005:547792  CAPLUS
DOCUMENT NUMBER:          143:76842
TITLE:                    Macrophage inflammatory protein-4 (MIP-
                          4) as an endogenous ligand for CCRL2
                          and sequences of human MIP-4 and
                          CCRL2
INVENTOR(S):              Tinsley, Jonathon Mark
PATENT ASSIGNEE(S):       Oxagen Limited, UK
SOURCE:                   PCT Int. Appl., 69 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.           KIND   DATE        APPLICATION NO.         DATE
     ---------------      ----   --------    --------------------    --------
     WO 2005057220         A2    20050623    WO 2004-GB5057          20041202
     WO 2005057220         A3    20060202
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
             CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
             GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
             LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,
             NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
             TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
         RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM,
             AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK,
             EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT,
             RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML,
             MR, NE, SN, TD, TG
     EP 1692171            A2    20060823    EP 2004-801256          20041202
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK,
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BA, HR, IS, YU
JP 2007520210 T 20070726 JP 2006-542005 20041202
US 2007036781 A1 20070215 US 2006-579386 20060515
PRIORITY APPLN. INFO.: GB 2003-28275 A 20031205
GB 2004-3014 A 20040211
GB 2004-18568 A 20040819
WO 2004-GB5057 W 20041202

AB Macrophage inflammatory protein-4 (MIP-4; also known as DC-CKI, CCL18 and PARC) is identified as an endogenous ligand for CCRL2 (chemokine (C-C motif) receptor-like 2). The protein and cDNA sequences of human MIP-4 and CCRL2 are disclosed. Anti-CCRL2 antibody was blocking MIP-4 and synovial fluid induced monocyte chemotaxis. Anti-MIP-4 antibody was also blocking RA synovial fluid induced monocyte chemotaxis. This data demonstrates that MIP-4 is a major mediator of monocyte induced chemotaxis found in RA synovial fluid. CCRL2 modulators, such as antibodies against CCRL2 or MIP-4, is useful in treating an inflammatory disease, a disease associated with enhanced macrophage activity or an infection.

=> S Antibody(S)MIP-4 AND pd<=20041202
2 FILES SEARCHED...
L2 1 ANTIBODY(S) MIP-4 AND PD<=20041202

=> D ibib abs L2

L2 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1995:820779 CAPLUS
DOCUMENT NUMBER: 123:220290
TITLE: Cloning and therapeutic applications of human macrophage inflammatory proteins MIP-3, MIP-4, and MIP-1γ, or their antibodies or antagonists
INVENTOR(S): Li, Haodong; Rosen, Craig A.; Ruben, Steve; Adams, Mark D.
PATENT ASSIGNEE(S): Human Genome Sciences, Inc., USA
SOURCE: PCT Int. Appl., 63 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 10
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9517092	A1	19950629	WO 1994-US7256	19940628 <--
W: AU, CA, CN, JP, KR, NZ				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 5556767	A	19960917	US 1993-173209	19931222 <--
US 5504003	A	19960402	US 1994-208339	19940308 <--
ZA 9403442	A	19951120	ZA 1994-3442	19940518 <--
CA 2179606	A1	19950629	CA 1994-2179606	19940628 <--
AU 9475497	A	19950710	AU 1994-75497	19940628 <--
AU 684539	B2	19971218		
EP 735818	A1	19961009	EP 1994-925671	19940628 <--
EP 735818	B1	20040331		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
CN 1143894	A	19970226	CN 1994-194902	19940628 <--
JP 09506774	T	19970708	JP 1995-517397	19940628 <--
JP 3677288	B2	20050727		

JP 2002053490	A	20020219	JP 2001-196723		19940628 <--
AT 262914	T	20040415	AT 1994-925671		19940628 <--
PT 735818	T	20040730	PT 1994-925671		19940628 <--
ES 2214484	T3	20040916	ES 1994-925671		19940628 <--
CN 1321745	A	20011114	CN 2001-116577		20010416 <--
AU 777297	B2	20041007	AU 2002-15445		20020206 <--
US 2003147846	A1	20030807	US 2002-165233		20020610 <--
PRIORITY APPLN. INFO.:			US 1993-173209	A	19931222
			US 1994-208339	A	19940308
			JP 1995-517397	A3	19940628
			WO 1994-US7256	W	19940628
			US 1995-446881	B1	19950505
			US 1995-468775	B2	19950606
			AU 1997-46576	A3	19970930
			US 1999-334923	A3	19990617
			US 1999-334951	A3	19990617
			US 1999-334954	A3	19990617

AB There are disclosed human macrophage inflammatory protein-3, human macrophage inflammatory protein-4, and human macrophage inflammatory protein-1γ polypeptides and DNA (or RNA) encoding such polypeptides.

There is also provided a procedure for producing such polypeptides by recombinant techniques and for producing antibodies against such polypeptides. In the invention there is also provided antagonist/inhibitors against such polypeptides which inhibit the functioning of such polypeptides. Another aspect of the invention provides a combination of the polypeptides of the present invention and a suitable pharmaceutical carrier for providing a therapeutically effective amount of the polypeptides for the treatment of various associated diseases.

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=> S Antibody(S)CCRL-2 AND pd<=20041202
   2 FILES SEARCHED...
L3           0 ANTIBODY(S) CCRL-2 AND PD<=20041202

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 NEWS  3  AUG 06  FSTA enhanced with new thesaurus edition
 NEWS  4  AUG 13  CA/CAplus enhanced with additional kind codes for granted
                  patents
 NEWS  5  AUG 20  CA/CAplus enhanced with CAS indexing in pre-1907 records
 NEWS  6  AUG 27  Full-text patent databases enhanced with predefined
                  patent family display formats from INPADOCDB
 NEWS  7  AUG 27  USPATOLD now available on STN
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 NEWS  8  AUG 28  CAS REGISTRY enhanced with additional experimental
                  spectral property data
 NEWS  9  SEP 07  STN AnaVist, Version 2.0, now available with Derwent
                  World Patents Index
 NEWS 10  SEP 13  FORIS renamed to SOFIS
 NEWS 11  SEP 13  INPADOCDB enhanced with monthly SDI frequency
 NEWS 12  SEP 17  CA/CAplus enhanced with printed CA page images from
                  1967-1998
 NEWS 13  SEP 17  CAplus coverage extended to include traditional medicine
                  patents
 NEWS 14  SEP 24  EMBASE, EMBAL, and LEMBASE reloaded with enhancements
 NEWS 15  OCT 02  CA/CAplus enhanced with pre-1907 records from Chemisches
                  Zentralblatt
 NEWS 16  OCT 19  BEILSTEIN updated with new compounds
 NEWS 17  NOV 15  Derwent Indian patent publication number format enhanced
 NEWS 18  NOV 19  WPIX enhanced with XML display format
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 NEWS 20  DEC 04  LINPADOCDB now available on STN
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 NEWS 22  DEC 17  USPATOLD added to additional database clusters
 NEWS 23  DEC 17  IMSDRUGCONF removed from database clusters and STN
 NEWS 24  DEC 17  DGENE now includes more than 10 million sequences
 NEWS 25  DEC 17  TOXCENTER enhanced with 2008 MeSH vocabulary in
                  MEDLINE segment
 NEWS 26  DEC 17  MEDLINE and LMEDLINE updated with 2008 MeSH vocabulary
 NEWS 27  DEC 17  CA/CAplus enhanced with new custom IPC display formats
 NEWS 28  DEC 17  STN Viewer enhanced with full-text patent content
                  from USPATOLD
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 NEWS 30  JAN 16  CAS patent coverage enhanced to include exemplified
                  prophetic substances
 NEWS 31  JAN 28  USPATFULL, USPAT2, and USPATOLD enhanced with new
                  custom IPC display formats
 NEWS 32  JAN 28  MARPAT searching enhanced
 NEWS 33  JAN 28  USGENE now provides USPTO sequence data within 3 days
                  of publication
 NEWS 34  JAN 28  TOXCENTER enhanced with reloaded MEDLINE segment
 NEWS 35  JAN 28  MEDLINE and LMEDLINE reloaded with enhancements
 NEWS 36  FEB 08  STN Express, Version 8.3, now available


 NEWS EXPRESS FEBRUARY 08 CURRENT WINDOWS VERSION IS V8.3,
              AND CURRENT DISCOVER FILE IS DATED 24 JANUARY 2008

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                                                         ENTRY    SESSION
FULL ESTIMATED COST                                       0.21       0.21

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Ckbeta1 OR Ckbeta7) AND (CCRL2 OR HCR OR CRAM-A) AND pd<=20041202
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L1            6 (MIP-4 OR CCL18 OR PARC OR AMAC1 OR AMAC-1 OR DCCK1 OR DC-CK-1
                OR SCYA18 OR CKBETA1 OR CKBETA7) AND (CCRL2 OR HCR OR CRAM-A)
                AND PD<=20041202

=> Dup Rem L1
PROCESSING COMPLETED FOR L1
L2            2 DUP REM L1 (4 DUPLICATES REMOVED)
                ANSWERS '1-2' FROM FILE MEDLINE

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L2   ANSWER 1 OF 2     MEDLINE on STN                    DUPLICATE 1
ACCESSION NUMBER:   2004617243     MEDLINE
DOCUMENT NUMBER:    PubMed ID: 15588486
TITLE:              Haplotype structure and linkage disequilibrium in chemokine
                    and chemokine receptor genes.
AUTHOR:             Clark Vanessa J; Dean Michael
CORPORATE SOURCE:   Laboratory of Genomic Diversity, Human Genetics Section,
                    National Cancer Institute, Frederick, MD 21702, USA..
                    vclark@genetics.bsd.uchicago.edu
SOURCE:             Human genomics, (2004 May) Vol. 1, No. 4, pp.
                    255-73.
                    Journal code: 101202210. ISSN: 1473-9542.
PUB. COUNTRY:       England: United Kingdom
DOCUMENT TYPE:      Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:           English
FILE SEGMENT:       Priority Journals
ENTRY MONTH:        200503
ENTRY DATE:         Entered STN: 20 Dec 2004
                    Last Updated on STN: 30 Mar 2005
                    Entered Medline: 29 Mar 2005
AB   To dissect the haplotype structure of candidate genes for disease
     association studies, it is important to understand the nature of genetic
     variation at these loci in different populations.  We present a survey of
     haplotype structure and linkage disequilibrium of chemokine and chemokine
     receptor genes in 11 geographically-distinct population samples (n=728).
     Chemokine proteins are involved in intercellular signalling and the immune
     response.  These molecules are important modulators of human
     immunodeficiency virus (HIV)-1 infection and the progression of the
     acquired immune deficiency syndrome, tumour development and the metastatic
     process of cancer.  To study the extent of genetic variation in this gene
     family, single nucleotide polymorphisms (SNPs) from 13 chemokine and
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chemokine receptor genes were genotyped using the 5' nuclease assay (TaqMan). SNP haplotypes, estimated from unphased genotypes using the Expectation-Maximization-algorithm, are described in a cluster of four CC-chemokine receptor genes (CCR3, CCR2, CCR5 and CCRL2) on chromosome 3p21, and a cluster of three CC-chemokine genes [MPIF-1 (CCL23), PARC (CCL18) and MIP-1alpha (CCL3)] on chromosome 17q11-12. The 32 base pair (bp) deletion in exon 4 of CCR5 was also included in the haplotype analysis of 3p21. A total of 87.5 per cent of the variation of 14 biallelic loci scattered over 150 kilobases of 3p21 is explained by 11 haplotypes which have a frequency of at least 1 per cent in the total sample. An analysis of haplotype blocks in this region indicates recombination between CCR2 and CCR5, although long-range pairwise linkage disequilibrium across the region appears to remain intact on two common haplotypes. A reduced-median network demonstrates a clear relationship between 3p21 haplotypes, rooted by the putative ancestral haplotype determined by direct sequencing of four primate species. Analysis of six SNPs on 17q11-12 indicates that 97.5 per cent of the variation is explained by 15 haplotypes, representing at least 1 per cent of the total sample. Additionally, a possible signature of selection at a non-synonymous coding SNP (M106V) in the MPIF-1 (CCL23) gene warrants further study. We anticipate that the results of this study of chemokine and chemokine receptor variation will be applicable to more extensive surveys of long-range haplotype structure in these gene regions and to association studies of HIV-1 disease and cancer.

L2 ANSWER 2 OF 2 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2004617237 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15588479
TITLE: Characterisation of SNP haplotype structure in chemokine and chemokine receptor genes using CEPH pedigrees and statistical estimation.
AUTHOR: Clark Vanessa J; Dean Michael
CORPORATE SOURCE: Laboratory of Genomic Diversity, Human Genetics Section, National Cancer Institute, Frederick, MD 21702, USA.. vclark@genetics.bsd.uchicago.edu
SOURCE: Human genomics, (2004 Mar) Vol. 1, No. 3, pp. 195-207.
Journal code: 101202210. ISSN: 1473-9542.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200501
ENTRY DATE: Entered STN: 20 Dec 2004
Last Updated on STN: 19 Jan 2005
Entered Medline: 18 Jan 2005

AB Chemokine signals and their cell-surface receptors are important modulators of HIV-1 disease and cancer. To aid future case/control association studies, aim to further characterise the haplotype structure of variation in chemokine and chemokine receptor genes. To perform haplotype analysis in a population-based association study, haplotypes must be determined by estimation, in the absence of family information or laboratory methods to establish phase. Here, test the accuracy of estimates of haplotype frequency and linkage disequilibrium by comparing estimated haplotypes generated with the expectation maximisation (EM) algorithm to haplotypes determined from Centre d'Etude Polymorphisme Humain (CEPH) pedigree data. To do this, they have characterised haplotypes comprising alleles at 11 biallelic loci in four chemokine receptor genes (CCR3, CCR2, CCR5 and CCRL2), which span 150 kb on chromosome 3p21, and haplotyes of nine biallelic loci in six chemokine genes [MCP-1(CCL2), Eotaxin(CCL11), RANTES(CCL5), MPIF-1(CCL23), PARC(CCL18) and MIP-1alpha(CCL3)] on chromosome

17q11-12. Forty multi-generation CEPH families, totalling 489 individuals, were genotyped by the TaqMan 5'-nuclease assay. Phased haplotypes and haplotypes estimated from unphased genotypes were compared in 103 grandparents who were assumed to have mated at random.For the 3p21 single nucleotide polymorphism (SNP) data, haplotypes determined by pedigree analysis and haplotypes generated by the EM algorithm were nearly identical. Linkage disequilibrium, measured by the D' statistic, was nearly maximal across the 150 kb region, with complete disequilibrium maintained at the extremes between CCR3-Y17Y and CCRL2-I243V. D'-values calculated from estimated haplotypes on 3p21 had high concordance with pairwise comparisons between pedigree-phased chromosomes. Conversely, there was less agreement between analyses of haplotype frequencies and linkage disequilibrium using estimated haplotypes when compared with pedigree-phased haplotypes of SNPs on chromosome 17q11-12. These results suggest that, while estimations of haplotype frequency and linkage disequilibrium may be relatively simple in the 3p21 chemokine receptor cluster in population samples, the more complex environment on chromosome 17q11-12 will require a higher resolution haplotype analysis.

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NEWS 11	SEP 13	INPADOCDB enhanced with monthly SDI frequency
NEWS 12	SEP 17	CA/CAplus enhanced with printed CA page images from 1967-1998
NEWS 13	SEP 17	CAplus coverage extended to include traditional medicine patents
NEWS 14	SEP 24	EMBASE, EMBAL, and LEMBASE reloaded with enhancements
NEWS 15	OCT 02	CA/CAplus enhanced with pre-1907 records from Chemisches Zentralblatt
NEWS 16	OCT 19	BEILSTEIN updated with new compounds

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                  of publication
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FULL ESTIMATED COST                                       0.21       0.21

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=> S MIP-4 (S)CCRL2
L1 1 MIP-4 (S) CCRL2

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L1 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2008 ACS on STN
AB Macrophage inflammatory protein-4 (MIP-4; also known as DC-CKI, CCL18 and PARC) is identified as an endogenous ligand for CCRL2 (chemokine (C-C motif) receptor-like 2). The protein and cDNA sequences of human MIP-4 and CCRL2 are disclosed. Anti-CCRL2 antibody was blocking MIP-4 and synovial fluid induced monocyte chemotaxis. Anti-MIP-4 antibody was also blocking RA synovial fluid induced monocyte chemotaxis. This data demonstrates that MIP-4 is a major mediator of monocyte induced chemotaxis found in RA synovial fluid. CCRL2 modulators, such as antibodies against CCRL2 or MIP-4, is useful in treating an inflammatory disease, a disease associated with enhanced macrophage activity or an infection.

=> D Ibib

L1 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2005:547792 CAPLUS
DOCUMENT NUMBER: 143:76842
TITLE: Macrophage inflammatory protein-4 (MIP-4) as an endogenous ligand for CCRL2 and sequences of human MIP-4 and CCRL2
INVENTOR(S): Tinsley, Jonathon Mark
PATENT ASSIGNEE(S): Oxagen Limited, UK
SOURCE: PCT Int. Appl., 69 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005057220	A2	20050623	WO 2004-GB5057	20041202
WO 2005057220	A3	20060202		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1692171	A2	20060823	EP 2004-801256	20041202
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, BA, HR, IS, YU				
JP 2007520210	T	20070726	JP 2006-542005	20041202
US 2007036781	A1	20070215	US 2006-579386	20060515

PRIORITY APPLN. INFO.: GB 2003-28275 A 20031205
GB 2004-3014 A 20040211
GB 2004-18568 A 20040819
WO 2004-GB5057 W 20041202

=> S MIP-4 (S) receptor
L2 4 MIP-4 (S) RECEPTOR

=> Dup Rem L2
PROCESSING COMPLETED FOR L2
L3 4 DUP REM L2 (0 DUPLICATES REMOVED)
ANSWER '1' FROM FILE BIOSIS
ANSWERS '2-4' FROM FILE CAPLUS

=> D Ibib abs L3 1-4

L3 ANSWER 1 OF 4 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
ACCESSION NUMBER: 2007:307109 BIOSIS
DOCUMENT NUMBER: PREV200700295995
TITLE: Histamine release from the basophils of control and asthmatic subjects and a comparison of gene expression between "releaser". and "nonreleaser" basophils.
AUTHOR(S): Youssef, Lama A.; Schuyler, Mark; Gilmartin, Laura; Pickett, Gavin; Bard, Julie D. J.; Tarleton, Christy A.; Archibeque, Tereassa; Qualls, Clifford; Wilson, Bridget S.; Oliver, Janet M. [Reprint Author]
CORPORATE SOURCE: Univ New Mexico, Sch Med, Dept Cell Pathol Lab, 2325 Camino de Salud, Albuquerque, NM 87131 USA
joliver@salud.umn.edu
SOURCE: Journal of Immunology, (APR 1 2007) Vol. 178, No. 7, pp. 4584-4594.
CODEN: JOIMA3. ISSN: 0022-1767.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 9 May 2007
Last Updated on STN: 9 May 2007

AB Most human blood basophils respond to Fc epsilon RI cross-linking by releasing histamine and other inflammatory mediators. Basophils that do not degranulate after anti-IgE challenge, known as "nonreleaser" basophils, characteristically have no or barely detectable levels of the Syk tyrosine kinase. The true incidence of the nonreleaser phenotype, its relationship (if any) to allergic asthma, and its molecular mechanism are not well understood. In this study, we report statistical analyses of degranulation assays performed in 68 control and 61 asthmatic subjects that establish higher basal and anti-IgE-stimulated basophil degranulation among the asthmatics. Remarkably, 28% of the, control group and 13% of the asthmatic group were nonreleasers; for all or part of our 4-year long study and cycling between the releaser and nonreleaser phenotypes occurred at least once in blood basophils from 8 (of 8) asthmatic and 16 (of 23) control donors. Microarray analysis showed that basal gene expression was generally lower in nonreleaser than releaser basophils. In releaser I cells, Fc epsilon RI cross-linking up-regulated > 200 genes, including genes encoding receptors (the Fc epsilon RI a and beta subunits, the histamine 4 receptor, the chemokine (C-C motif) receptor 1), signaling proteins (Lyn), chemokines (IL-8, RANTES, MIP-1 alpha, and, MIP-4 beta) and transcription factors (early growth response-1, early growth response-3, and AP-1). Fc epsilon RI cross-linking induced fewer, and quite distinct, transcriptional responses in nonreleaser cells. We conclude that "nonreleaser" and "cycler" basophils represent a distinct and reversible natural phenotype. Although histamine is more readily released from

basophils isolated from asthmatics than controls, the presence of noureleaser basophils does not rule out the diagnosis of asthma.

L3 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2005:547792 CAPLUS
DOCUMENT NUMBER: 143:76842
TITLE: Macrophage inflammatory protein-4 (MIP-4) as an endogenous ligand for CCRL2 and sequences of human MIP-4 and CCRL2
INVENTOR(S): Tinsley, Jonathon Mark
PATENT ASSIGNEE(S): Oxagen Limited, UK
SOURCE: PCT Int. Appl., 69 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005057220	A2	20050623	WO 2004-GB5057	20041202
WO 2005057220	A3	20060202		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1692171	A2	20060823	EP 2004-801256	20041202
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, BA, HR, IS, YU				
JP 2007520210	T	20070726	JP 2006-542005	20041202
US 2007036781	A1	20070215	US 2006-579386	20060515

PRIORITY APPLN. INFO.:

GB 2003-28275	A	20031205
GB 2004-3014	A	20040211
GB 2004-18568	A	20040819
WO 2004-GB5057	W	20041202

AB Macrophage inflammatory protein-4 (MIP-4; also known as DC-CKI, CCL18 and PARC) is identified as an endogenous ligand for CCRL2 (chemokine (C-C motif) receptor-like 2). The protein and cDNA sequences of human MIP-4 and CCRL2 are disclosed. Anti-CCRL2 antibody was blocking MIP-4 and synovial fluid induced monocyte chemotaxis. Anti-MIP-4 antibody was also blocking RA synovial fluid induced monocyte chemotaxis. This data demonstrates that MIP-4 is a major mediator of monocyte induced chemotaxis found in RA synovial fluid. CCRL2 modulators, such as antibodies against CCRL2 or MIP-4, is useful in treating an inflammatory disease, a disease associated with enhanced macrophage activity or an infection.

L3 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2000:82601 CAPLUS
DOCUMENT NUMBER: 132:221179
TITLE: C-C chemokine receptor 3 antagonism by the β-chemokine macrophage inflammatory protein 4, a property strongly enhanced by an amino-terminal alanine-methionine swap

AUTHOR(S): Nibbs, Robert J. B.; Salcedo, Theodora W.; Campbell, John D. M.; Yao, Xiao-Tao; Li, Yuling; Nardelli, Bernardetta; Olsen, Henrik S.; Morris, Tina S.; Proudfoot, Amanda E. I.; Patel, Vikram P.; Graham, Gerard J.
CORPORATE SOURCE: Cancer Research Campaign Laboratories, Beatson Institute for Cancer Research, Glasgow, G61 1BD, UK
SOURCE: Journal of Immunology (2000), 164(3), 1488-1497
CODEN: JOIMA3; ISSN: 0022-1767
PUBLISHER: American Association of Immunologists
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Allergic reactions are characterized by the infiltration of tissues by activated eosinophils, Th2 lymphocytes, and basophils. The β-chemokine receptor CCR3, which recognizes the ligands eotaxin, eotaxin-2, monocyte chemotactic protein (MCP) 3, MCP4, and RANTES, plays a central role in this process, and antagonists to this receptor could have potential therapeutic use in the treatment of allergy. The authors describe here a potent and specific CCR3 antagonist, called Met-chemokine β 7 (Ckβ7), that prevents signaling through this receptor and, at concns. as low as 1 nM, can block eosinophil chemotaxis induced by the most potent CCR3 ligands. Met-Ckβ7 is a more potent CCR3 antagonist than Met- and aminooxypentane (AOP)-RANTES and, unlike these proteins, exhibits no partial agonist activity and is highly specific for CCR3. This antagonist may thus be of use in ameliorating leukocyte infiltration associated with allergic inflammation. Met-Ckβ7 is a modified form of the β-chemokine macrophage inflammatory protein (MIP) 4 [alternatively called pulmonary and activation-regulated chemokine (PARC), alternative macrophage activation-associated C-C chemokine (AMAC) 1, or dendritic cell-derived C-C chemokine (DCCK) 1]. Surprisingly, the unmodified MIP4 protein, which is known to act as a T cell chemoattractant, also exhibits this CCR3 antagonistic activity, although to a lesser extent than Met-Ckβ7, but to a level that may be of physiol. relevance. MIP4 may therefore use chemokine receptor agonism and antagonism to control leukocyte movement in vivo. The enhanced activity of Met-Ckβ7 is due to the alteration of the extreme N-terminal residue from an alanine to a methionine.

REFERENCE COUNT: 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 1995:820779 CAPLUS
DOCUMENT NUMBER: 123:220290
TITLE: Cloning and therapeutic applications of human macrophage inflammatory proteins MIP-3, MIP-4, and MIP-1γ, or their antibodies or antagonists
INVENTOR(S): Li, Haodong; Rosen, Craig A.; Ruben, Steve; Adams, Mark D.
PATENT ASSIGNEE(S): Human Genome Sciences, Inc., USA
SOURCE: PCT Int. Appl., 63 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 10
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9517092	A1	19950629	WO 1994-US7256	19940628
W: AU, CA, CN, JP, KR, NZ				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 5556767	A	19960917	US 1993-173209	19931222

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     US 5504003           A     19960402    US 1994-208339         19940308
     ZA 9403442           A     19951120    ZA 1994-3442           19940518
     CA 2179606           A1    19950629    CA 1994-2179606        19940628
     AU 9475497           A     19950710    AU 1994-75497          19940628
     AU 684539            B2    19971218
     EP 735818            A1    19961009    EP 1994-925671         19940628
     EP 735818            B1    20040331
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE
     CN 1143894           A     19970226    CN 1994-194902         19940628
     JP 09506774          T     19970708    JP 1995-517397         19940628
     JP 3677288           B2    20050727
     JP 2002053490        A     20020219    JP 2001-196723         19940628
     AT 262914            T     20040415    AT 1994-925671         19940628
     PT 735818            T     20040730    PT 1994-925671         19940628
     ES 2214484           T3    20040916    ES 1994-925671         19940628
     CN 1321745           A     20011114    CN 2001-116577         20010416
     AU 777297            B2    20041007    AU 2002-15445          20020206
     US 2003147846        A1    20030807    US 2002-165233         20020610
PRIORITY APPLN. INFO.:                      US 1993-173209      A  19931222
                                            US 1994-208339      A  19940308
                                            JP 1995-517397      A3 19940628
                                            WO 1994-US7256      W  19940628
                                            US 1995-446881      B1 19950505
                                            US 1995-468775      B2 19950606
                                            AU 1997-46576       A3 19970930
                                            US 1999-334923      A3 19990617
                                            US 1999-334951      A3 19990617
                                            US 1999-334954      A3 19990617
AB   There are disclosed human macrophage inflammatory protein-3, human
     macrophage inflammatory protein-4, and human macrophage inflammatory
     protein-1γ polypeptides and DNA (or RNA) encoding such polypeptides.
     There is also provided a procedure for producing such polypeptides by
     recombinant techniques and for producing antibodies against such
     polypeptides.  In the invention there is also provided
     antagonist/inhibitors against such polypeptides which inhibit the
     functioning of such polypeptides.  Another aspect of the invention
     provides a combination of the polypeptides of the present invention and a
     suitable pharmaceutical carrier for providing a therapeutically effective
     amount of the polypeptides for the treatment of various associated diseases.


=> S CCRL2 (S) ligand
L4           5 CCRL2 (S) LIGAND

=> Dup Rem L4
PROCESSING COMPLETED FOR L4
L5             3 DUP REM L4 (2 DUPLICATES REMOVED)
                 ANSWER '1' FROM FILE MEDLINE
                 ANSWER '2' FROM FILE BIOSIS
                 ANSWER '3' FROM FILE CAPLUS

=> D Ibib Abs L5 1-3

L5   ANSWER 1 OF 3     MEDLINE on STN                    DUPLICATE 1
ACCESSION NUMBER:   2004286185     MEDLINE
DOCUMENT NUMBER:    PubMed ID: 15188357
TITLE:              Up-regulated expression and activation of the orphan
                    chemokine receptor, CCRL2, in rheumatoid arthritis.
AUTHOR:             Galligan Carole L; Matsuyama Wataru; Matsukawa Akihiro;
                    Mizuta Hiroshi; Hodge David R; Howard O M Zack; Yoshimura
                    Teizo
CORPORATE SOURCE:   National Cancer Institute at Frederick, Frederick, Maryland
```

21702, USA.
SOURCE: Arthritis and rheumatism, (2004 Jun) Vol. 50, No. 6, pp. 1806-14.
Journal code: 0370605. ISSN: 0004-3591.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200407
ENTRY DATE: Entered STN: 10 Jun 2004
Last Updated on STN: 9 Jul 2004
Entered Medline: 8 Jul 2004

AB OBJECTIVE: Rheumatoid arthritis (RA) is a chronic inflammatory condition characterized by a cellular influx and destruction of the joint architecture. Chemokines characteristically regulate leukocyte recruitment and activation. Chemokine (CC motif) receptor-like 2 (CCRL2) is an orphan receptor with homology to other CC chemokine receptors. We undertook this study to examine CCRL2 expression in RA, cytokine regulation of expression, and the source of a putative ligand in an attempt to determine the role of this receptor during inflammation. METHODS: Expression of CCRL2 on joint-infiltrating leukocytes was examined by immunocytochemistry. In vitro studies evaluated CCRL2 expression in primary neutrophils using Northern and Western blotting and reverse transcriptase-polymerase chain reaction. HEK 293 cells expressing two splice variants of CCRL2 (HEK/CCRL2A or HEK/CCRL2B) were generated with a retroviral expression system, and their migration in response to fractions of synovial fluid (SF) from RA patients was examined using a 48-well chamber. RESULTS: CCRL2 expression was observed on all infiltrating neutrophils and on some macrophages obtained from the SF of 5 RA patients. In vitro studies of primary neutrophils revealed that CCRL2 messenger RNA (mRNA) was rapidly up-regulated following stimulation with lipopolysaccharide (1 microg/ml) or tumor necrosis factor (5 ng/ml). The mRNA for both CCRL2A and CCRL2B were expressed in cytokine-stimulated neutrophils. Cells expressing either of these splice variants migrated in response to a fraction of RA SF. CONCLUSION: CCRL2 expression is up-regulated on synovial neutrophils of RA patients. Inflammatory products present in the SF activate this receptor, indicating that CCRL2 is a functional receptor that may be involved in the pathogenesis of RA.

L5 ANSWER 2 OF 3 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:286307 BIOSIS
DOCUMENT NUMBER: PREV200400285064
TITLE: Upregulated expression and activation of the orphan chemokine receptor, CCRL2, in rheumatoid arthritis.
AUTHOR(S): Galligan, Carole [Reprint Author]; Matsuyama, Wataru; Matsukawa, Akihiro; Mizuta, Hiroshi; Hodge, David R; Howard, O.M. Zack; Yoshimura, Teizo
CORPORATE SOURCE: Laboratory of Molecular Immunoregulation, National Cancer Institute, P.O. Box B, Bldg. 560, Frederick, MD, 21702-1201, USA
cgalligan@ncifcrf.gov
SOURCE: FASEB Journal, (2004) Vol. 18, No. 4-5, pp. Abst. 337.9.
http://www.fasebj.org/. e-file.
Meeting Info.: FASEB Meeting on Experimental Biology: Translating the Genome. Washington, District of Columbia, USA. April 17-21, 2004. FASEB.
ISSN: 0892-6638 (ISSN print).
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 16 Jun 2004
Last Updated on STN: 16 Jun 2004

AB Rheumatoid arthritis (RA) is a chronic inflammatory condition characterized by a cellular influx and destruction of the joint architecture involving chemokines that induce the leukocyte infiltration and activation. The human chemokine-like receptor 2 (CCRL2) codes for a putative 7-TM G protein-coupled receptor with high homology to other chemokine receptors. This study examined CCRL2 expression in RA, the cytokines regulating gene expression and the source of a putative ligand for CCRL2 in an attempt to determine the role of this receptor during inflammation. Immunohistochemistry revealed positive CCRL2 staining of neutrophils infiltrating the joints of RA patients. Primary human neutrophils expressed low levels of CCRL2 mRNA, but stimulation with LPS or TNF dramatically upregulated mRNA levels. Elevated CCRL2 mRNA expression was evident as early as 1 h after TNF- or LPS-activation and the levels peaked after 2-4 or 4-8 hours respectively. Two N-terminal splice variants for CCRL2 (A and B) were detected in freshly isolated as well as in LPS- and TNF-activated neutrophils by RT-PCR. CCRL2 protein was not detectable in freshly isolated neutrophils but readily detectable in LPS-activated neutrophils. Fractions of RA synovial fluids induced significant chemotaxis for HEK-293 cells expressing either CCRL2 variant. Our results suggest that CCRL2 may play a role in regulating neutrophil recruitment and activation during rheumatoid arthritis.

L5 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2005:547792 CAPLUS
DOCUMENT NUMBER: 143:76842
TITLE: Macrophage inflammatory protein-4 (MIP-4) as an endogenous ligand for CCRL2 and sequences of human MIP-4 and CCRL2
INVENTOR(S): Tinsley, Jonathon Mark
PATENT ASSIGNEE(S): Oxagen Limited, UK
SOURCE: PCT Int. Appl., 69 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005057220	A2	20050623	WO 2004-GB5057	20041202
WO 2005057220	A3	20060202		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1692171	A2	20060823	EP 2004-801256	20041202
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, BA, HR, IS, YU				
JP 2007520210	T	20070726	JP 2006-542005	20041202
US 2007036781	A1	20070215	US 2006-579386	20060515
PRIORITY APPLN. INFO.:			GB 2003-28275 A	20031205
			GB 2004-3014 A	20040211
			GB 2004-18568 A	20040819

WO 2004-GB5057 W 20041202

AB Macrophage inflammatory protein-4 (MIP-4; also known as DC-CKI, CCL18 and PARC) is identified as an endogenous ligand for CCRL2 (chemokine (C-C motif) receptor-like 2). The protein and cDNA sequences of human MIP-4 and CCRL2 are disclosed. Anti-CCRL2 antibody was blocking MIP-4 and synovial fluid induced monocyte chemotaxis. Anti-MIP-4 antibody was also blocking RA synovial fluid induced monocyte chemotaxis. This data demonstrates that MIP-4 is a major mediator of monocyte induced chemotaxis found in RA synovial fluid. CCRL2 modulators, such as antibodies against CCRL2 or MIP-4, is useful in treating an inflammatory disease, a disease associated with enhanced macrophage activity or an infection.

=> Log Off H
SESSION WILL BE HELD FOR 120 MINUTES
STN INTERNATIONAL SESSION SUSPENDED AT 20:19:13 ON 16 FEB 2008